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ORIGINAL ARTICLE

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Growth and dissipation in biological tissues

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Abstract This paper provides a unified mathematical framework so as to study the growth of biological tissues on an energetic basis. All the contributions to growth of solute chemicals and nutrients are here resumed in one scalar descriptor, the biochemical energy of the system. The free energy of the system accounts for both strain and biochemical storage. The exploitation of a dissipation inequality by standard means provides admissible couplings between growth, tension and energy. Specific admissible constitutive equations lead back, in some cases, to classical models.

Keywords Growth · Soft biological tissues · Energy inequality · Nutrient factors

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Introduction

In soft biological tissues, growth takes place at a rate that may be influenced by many factors, including genetical predisposition, mechanical stimuli and availability of nutrients. Even when restricting the field to the macroscopic scale only, the relevant scientific literature usually focuses just on one of these mechanisms at a time; some authors consider the mechanical aspects of growth, while others focus on the biochemical aspects associated with growth processes in terms of coupled reaction and diffusion. Most mathematical models developed for the description of growth in soft biological tissues, such as arteries, are based on a purely mechanical approach (see [15,18] for example). In these models, the mechanical stimulus generating growth (and possibly residual stresses) is assumed to dictate entirely the process, regardless of other factors, such as the availability of nutrients, which are instead shown to be of primary importance in the biochemical literature. Conversely, the dynamics of nutrients and growth factors is usually described in the biomathematical literature by a number of reaction-diffusion equations, linear in higher order terms and nonlinearly coupled by the non-differential right hand-side terms; nevertheless no energetic balance ever appears (see for example [13] and the huge reference list therein).

This paper is an attempt to design a framework that accounts for the role of both mechanical stimuli and biochemical factors in a rational way; the aim is to recover, as limit cases, standard reaction-diffusion equations for the chemical species as well as stress-modulated growth laws that are reported in the biomechanics literature, the coupling between these two mechanisms being thermodynamically admissible.

In the present work, we follow the definition by Taber [18] and we regard *growth* as a change in mass and geometry, while *remodelling* is taken to represent modifications in the mechanical properties of the material and is not considered here. The soft biological tissue is here represented as a single-component material, so that

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the growth model presented here is not based on mixture theory. Furthermore, instead of trying to characterize separately the various chemical species that are primordial to growth, we consider only one scalar quantity to represent them. This quantity appears within the model in the form of a so-called "biochemical" energy per unit mass, denoted by c, that represents the energy for growth available for creating new material within the tissue, i.e., new mass that will play an active part in the mechanical response of the tissue. By doing so we are able to focus on the crucial aspects of the problem while avoiding unnecessary complications.

On the basis of quite general assumptions and a rigorous derivation, we obtain a class of thermodynamically admissible growth laws coupling biochemical energy and tensional state. The predicted dynamics is in agreement with some of the phenomenological descriptions usually appearing in the literature.

The first section of the paper is devoted to a sketch of the biochemical processes underlying the growth dynamics. Basic notions of exchanges at a cellular level are briefly recalled with the related chemical reactions and the dependence of the growth rate on biochemical aspects is discussed. In Sect. 2 the balance equations for a single-component material subject to growth are presented. In particular, the energy balance includes the biochemical energy contribution associated with the growth. Admissible constitutive equations for growth are obtained in Sect. 3 after stating and exploiting a dissipation inequality. Simple growth laws involve a novel coupling between dynamics and biochemical factors. In the final section some implications of the present model are discussed.

1 Biochemical energy and growth

In a soft biological tissue, such as an artery, there are basically three mechanically important constituents, namely the smooth muscle cells, the elastin and the collagen. While smooth muscle cells, which are responsible for the viscoelastic behavior of arteries, grow primarily by hyperplasia (i.e., cell division) during development, hypertrophy (i.e., cell enlargement) is predominant at maturity [17]. In addition, collagen and elastin, which are two long-chained proteins, are produced by fibroblast cells and fibrocyte cells, respectively. Let us note further that, although collagen, which generates strong anisotropy in the arterial response, is produced throughout the whole life, nearly all the elastin present in an artery is created solely during development [10]. The growth of smooth muscle cells or the ability of fibroblasts and fibrocytes to produce collagen and elastin is directly linked to the availability of nutrients in the cells surrounding. In fact, in an arterial tissue, cells are constantly bathed in a substance ground matrix that contains, among other constituents, growth factors, proteases, ions, hormones, metabolites and nutrients. In particular, nutrients, which are originally supplied by the bloodstream, provide a source of rough material from which cells may draw in order to grow and sustain themselves.

Which mechanical quantity, such as stress, strain or strain rate, is to be held responsible for the appearance of growth and/or remodelling in arterial tissues is still a matter of debate among researchers, and experimental findings exist that seem to support each. For example, it is well acknowledged nowadays that growth in arteries is accompanied by a return of the average circumferential stress (see, for example, [11]) and of the average axial stress (see [10]) near uniform homeostatic values. This correlation between stress and growth seems to support stress over strain as the possible mechanical regulator for arterial growth and, in fact, it has served as a basis for a large amount of stress-related arterial growth models (see, [15,18] or [16], for example).

As a matter of fact, at a microscopic level, not only the exact mechanical stimulus triggering growth is largely unknown, but it is also not clear how the cells sense mechanical signals and convert them into chemical signals that will ultimately induce a biological response such as growth [19]. Most studies dealing with signal transduction (i.e., a change of signal from mechanical one to chemical one) focus on cellular membrane components, such as integrins and receptors for growth factors. These components on the cell surface are thought to act as mechanosensory entities [8], so that mechanical forces, by altering the cell surface, may directly perturb membrane component conformation, subsequently activating some signal transduction pathways for growth factors activity. In an interesting work on platelet-derived growth factor receptors from vascular smooth muscle cells, it has been suggested that mechanical forces might be absorbed by cell membrane receptors in the form of energy, thereby activating these receptors [9]. The subsequent binding of growth factors on these receptors then initiates an interlinked series of biochemical events inside the cell, involving enzymes, proteins and ions (especially calcium). In particular, growth factor proteins act as regulators for the transport of a wide variety of nutrients within the cell and can be roughly thought of as a "permission signal" for cells to access extracellular nutrients, therefore controlling cell growth and survival.

It is worth emphasizing that the succession of chemical events occurring inside a cell and leading to its growth is still very poorly understood, as most studies on the subject usually focus on the effects of one particular chemical component at a time, such a component often being originally located on the surface of the cell.

That is, the overall restricted knowledge of the different biochemical reactions involved in cell growth make it hard, if not impossible at the present time, to provide a macroscopic mechanical growth model that would account for all the different microscopical steps involved in signal transduction pathways.

In order to account for all these important biochemical events, which occur between the initial mechanical stimulus and the observed macroscopic growth of the tissue, here we introduce a single scalar quantity in the form of a biochemical energy, that would potentially reflect possible defects occurring in these biochemical processes that link the mechanical stimulus to the growth response. In particular, one of the aim of this quantity is to account, within the mechanical growth model, for the possibility of modelling an alteration in the overall biochemical process leading to growth that may be due to, for example, a low availability of nutrients in the cell surrounding. In fact, physically, if the nearby nutrients that serve as a source of rough material for cells to grow are at a dramatically low concentration, the cell will be unable to respond efficiently, even if there is a mechanical stimulus for growth acting on it. This will in turn affect the macroscopic growth rate associated with the tissue response. However, if the nutrients are widely available, the rate of biochemical reactions (and, subsequently, the growth rate), will not be bounded by nutrients concentration, but will be bounded by other factors, such as the mechanical stimulus that acts as a direct signal for growth to start.

An example of dependance of the growth rate on the biochemical energy can be recast in the popular Michaelis–Menten model [12]. In 1913 these authors proposed a simple kinetic law driving enzymatic chemical reactions. In the present work, we rephrase their enzimatic reaction rate depending on substrate concentration as the growth rate of the tissue depending on local biochemical energy concentration c. The growth rate Γ then reads

$$\Gamma = \Gamma_0 \frac{c}{K + c}, \tag{1.1}$$

where K is a constant and Γ_0 is the maximum growth rate. In the next the maximum growth rate will be assumed to be related to the mechanical growth stimulus, in a form to be determined. In the limit case in which the biochemical energy is widely available, a "completely" mechanically modulated growth law should be recovered.

2 Balance equations

In this section we summarize the balance equations, in Lagrangian form, that must be satisfied by a one-component continuum growing body. Let a material point of the body be labelled by its position vector $\mathbf{X}$ in the initial stress-free configuration, denoted $\mathcal{B}_0$, and by its position vector $\mathbf{x}$, at time t, in the current configuration, denoted $\mathcal{B}_t$. The deformation of the body from $\mathcal{B}_0$ to $\mathcal{B}_t$ may then characterized by a so-called deformation gradient, which is defined by $\mathbf{F} = \partial \mathbf{x}/\partial \mathbf{X}$.

In order to analyze the growth process, we adopt the multiplicative decomposition of the total deformation gradient, first introduced by Rodriguez et al. [14]. That is, the deformation gradient $\mathbf{F}$ is decomposed into a growth part, which characterizes possible changes occurring in the local stress-free state of the reference state, and an elastic part that ensures compatibility of the growth and also accounts for the loading process. The growth and the elastic components of the deformation are represented by the tensors $\mathbf{G}$ and $\mathbf{F}_r$, respectively. Thus, the multiplicative decomposition of the tensor gradient of deformation may be written as

$$\mathbf{F} = \mathbf{F}_r \mathbf{G}. \tag{2.1}$$

Let ρ denote the density of a material point at $\mathbf{X}$ and let $J = \det \mathbf{F}$. The growth within the body is assumed to occur solely volumetrically, and the volumetric growth rate per unit current mass is represented by Γ. The Lagrangian local form of the mass balance is then given by

$$\dot{(\rho J)} = \Gamma \rho J. \tag{2.2}$$

The growth tensor $\mathbf{G}$, whose form is dictated by a constitutive law (to be constitutively specified), is related to the volumetric growth rate Γ through [14]

$$\Gamma = \mathbf{I} \cdot \dot{\mathbf{G}} \mathbf{G}^{-1}, \tag{2.3}$$

where the upper dot corresponds to the material time derivative and $\mathbf{I}$ is the identity tensor.

The Lagrangian local form of the linear momentum balance is written as

$$\dot{(\rho J \mathbf{v})} = \rho J \mathbf{b} + \operatorname{Div} \mathbf{P}, \tag{2.4}$$

where $\mathbf{v}$ represents the velocity of a material point, $\mathbf{b}$ denotes the body forces, and $\mathbf{P}$ corresponds to the Piola–Kirchhoff stress. The inertia terms may reasonably be neglected, since growth is a very slow process (i.e., quasi-static deformations), and, in the absence of body forces, the linear momentum balance reduces to the equilibrium equation $\mathrm{Div}\,\mathbf{P} = \mathbf{0}$. In addition, for the angular momentum balance to be satisfied, the following relation must hold

$$\mathbf{F}\mathbf{P}^T = \mathbf{P}\mathbf{F}^T. \tag{2.5}$$

As mentioned beforehand, we introduce a specific form of energy associated with the growth process, namely the 'biochemical' energy. A crucial point is, therefore, to provide a balance equation for a generic solute carrier of the biochemical energy within the tissue that would account for the way this energy is spent in order to generate the growth.

Let us denote by $c(\mathbf{X}, t)$ the concentration per unit mass of a generic solute characterized by the surface flux $\mathbf{m}$ in a material frame of reference (to be constitutively specified) and by a bulk source (sink) spent for growth. Importantly, the form of the volumetric source (sink) of solute concentration is suggested by the growth kinematics, so that the dissipation rate of c for growth is taken to be proportional to the growth rate dictated by Eq. (2.3). Finally, the local balance equation of biochemical energy, in Lagrangian form, is expressed as

$$\rho J \dot{c} + \mathrm{Div}\,\mathbf{m} = \rho J \mathbf{E}_0 \cdot \dot{\mathbf{G}}\mathbf{G}^{-1}, \tag{2.6}$$

where $\mathbf{E}_0$ is some constant matrix accounting for the non-isotropic absorption rate. Directionality of production is related to the possible non-isotropic mechanical properties of the material. The vector $\mathbf{m}$ can be thought of as a mechanism for bringing nutrients that can be specified case by case as glucose, ATP, growth factors or other. The term on the right-hand side of Eq. (2.6) characterizes the rate of consumption (per unit reference volume) expelled by the body in order to generate the growth. According to Eq. (2.6), there is no convective transport of the solute c out of the motion of the body where it is released and therefore no convection is observed in a material frame of reference.

3 Constitutive theory

In this section we directly state and exploit a dissipative principle to be abided by any mechano-chemical process described by Eqs. (2.4) and (2.6).

Let us now introduce the free energy per unit mass ψ, defined as the portion of total energy stored by the system in reversible form. By neglecting the thermal terms contribution, we directly state the following dissipation inequality in integral form,

$$\frac{d}{\mathrm{d}t}\int_{\Omega} \rho J \psi \, d\Omega \leq \int_{\partial\Omega} \mathbf{P}\mathbf{n} \cdot \mathbf{v} \, d\Sigma - \int_{\partial\Omega} \mu \, \mathbf{m} \cdot \mathbf{n} \, d\Sigma. \tag{3.1}$$

According to the inequality (3.1), the energy supplied to the system in terms of mechanical work or biochemical flux is at most all stored in such a way that it can be recovered in the same form. The inequality must hold for any $\Omega \subset \mathcal{B}_o$ with outgoing normal $\mathbf{n}$. The scalar field μ is the chemical potential, which is provided to the system proportionally to the flux of solute $\mathbf{m}$.

The differential form of the inequality (3.1) is given by (compare with [4])

$$(\rho \dot{J} \psi) \leq \mathbf{P} \cdot \dot{\mathbf{F}} - \mathrm{Div}\ (\mu \mathbf{m}) \,. \tag{3.2}$$

In the present context the free energy ψ is taken to depend on the elastic part of the total deformation gradient $\mathbf{F}_r$ and on the solute concentration c, namely $\psi = \psi\,(\mathbf{F}_r, c)$.

Recalling Eqs. (2.1), (2.2) and (2.3), one may rewrite the inequality (3.1), after explicit derivation of the term at the left-hand side, as

$$\rho J \psi \, \mathbf{I} \cdot \dot{\mathbf{G}}\mathbf{G}^{-1} + \rho J \frac{\partial \psi}{\partial \mathbf{F}_r} \cdot \dot{\mathbf{F}}_r + \rho J \frac{\partial \psi}{\partial c} \dot{c} \leq \mathbf{P} \cdot (\dot{\mathbf{F}}_r \mathbf{G} + \mathbf{F}_r \dot{\mathbf{G}}) - \mu \mathrm{Div}\,\mathbf{m} - \mathbf{m} \cdot \mathrm{Grad}\,\mu. \tag{3.3}$$

The use of Eq. (2.6) in (3.3), together with a rearrangement of the various terms appearing in the resulting equation, yields

$$\begin{aligned} 0 \leq &\left(\mathbf{P}\mathbf{G}^T - \rho J \frac{\partial \psi}{\partial \mathbf{F}_r}\right) \cdot \dot{\mathbf{F}}_r - \left(\frac{\partial \psi}{\partial c} - \mu\right) \rho J \dot{c} - \mathbf{m} \cdot \operatorname{Grad} \mu \\ &+ \left(\mathbf{F}_r^T \mathbf{P}\mathbf{G}^T - \rho J \psi \mathbf{I} - \rho J \frac{\partial \psi}{\partial c} \mathbf{E}_0\right) \cdot \dot{\mathbf{G}}\mathbf{G}^{-1}. \end{aligned} \tag{3.4}$$

We require the inequality (3.4) to hold for any arbitrary motion, that is for any independent variation of $\mathbf{F}_r$ and c. This argument provides us with two constitutive equations:

$$\mathbf{P} = \rho J \frac{\partial \psi}{\partial \mathbf{F}_r} \mathbf{G}^{-T}, \tag{3.5}$$

$$\mu = \frac{\partial \psi}{\partial c}. \tag{3.6}$$

Equation (3.6) is the standard definition of chemical potential. Equation (3.5) relates the Piola–Kirchhoff stress tensor to the free energy. Note that differential form of the balance of forces cannot be stated in the intermediate configuration $\mathcal{B}_r$, where compatibility is lacking. It remains an unresolved question how the strain energy of the relaxed material can be measured, as such a relaxed configuration is never fully achieved by many soft biological tissues (see [7]). As a matter of fact, strain energies used in practice are obtained on the basis of tests performed on samples that have released most of the residual stress due to geometrical constraints.

The following residual inequality remains to be satisfied:

$$0 \leq -\mathbf{m} \cdot \operatorname{Grad} \mu + \left(\mathbf{F}_r^T \mathbf{P}\mathbf{G}^T - \rho J \psi \mathbf{I} - \rho J \mu \mathbf{E}_0\right) \cdot \dot{\mathbf{G}}\mathbf{G}^{-1}. \tag{3.7}$$

In particular, this requirement is always accomplished if

$$\begin{aligned} \left(\mathbf{F}_r^T \mathbf{P}\mathbf{G}^T - \rho J \psi \mathbf{I} - \rho J \mu \mathbf{E}_0\right) \cdot \dot{\mathbf{G}}\mathbf{G}^{-1} &\geq 0, \\ \mathbf{m} \cdot \operatorname{Grad} \mu &\leq 0. \end{aligned} \tag{3.8}$$

Remarkably, requiring that the two relationships (3.8) are satisfied does not yield constitutive equations, as in (3.5); they are instead dissipation-type inequalities that provide restrictions on the admissible form of the nutrient flow $\mathbf{m}$ and of the growth law giving the evolution in time of $\mathbf{G}$. The illustrated arguments suggest simple constitutive equations ensuring that the dissipation principle (3.1) is always satisfied. The flux of solute chemical can simply be taken as

$$\mathbf{m} = -\mathbf{K}_o \operatorname{Grad} \mu, \tag{3.9}$$

where $\mathbf{K}_o$ is a positive definite matrix.

For (3.8) to hold, a simple acceptable constitutive equation is

$$\mathbf{F}_r^T \mathbf{P}\mathbf{G}^T - \rho J \psi \mathbf{I} - \rho J \mu \mathbf{E}_0 = f(c)\, \dot{\mathbf{G}}\mathbf{G}^{-1}, \tag{3.10}$$

with $f(c)$ being a scalar non-negative function of the solute concentration c. By introducing the Eshelby tensor $\mathbf{E}$, defined as $\mathbf{E} = \mathbf{F}_r^T (\partial \psi / \partial \mathbf{F}_r) - \psi \mathbf{I}$, one may rewrite Eq. (3.10) as

$$\dot{\mathbf{G}}\mathbf{G}^{-1} = g(c) \rho J \left(\mathbf{E} - \mu \mathbf{E}_0\right), \tag{3.11}$$

where $g(c) = 1/f(c)$. Equation (3.10) is not a constitutive equation per se, but rather a necessary condition for the obtention of admissible constitutive equations that would describe the growth process. A reader familiar with plasticity theory might notice the analogy with the so-called "yield condition" (see, for example, [3]): the restriction (3.10) enables us to obtain a framework in which the number of equation equals the number of unknowns.

A simple choice for the function $g(c)$ is suggested by the biochemical processes involving growth at a cellular level and their apparent similitude with chemical reactions models, such as the one developed by Michaelis and Menten discussed in Sect. 1. Inspired by Eq. (1.1), a simple admissible choice for $g(c)$ is in fact

$$\dot{\mathbf{G}}\mathbf{G}^{-1} = \frac{c}{c_o + c} \rho J \left(\mathbf{E} - \mu \mathbf{E}_0\right), \tag{3.12}$$

where c_0 is a constant.

4 Discussion

In this paper biochemical and mechanical stimuli for growth are put in a unified thermodynamical framework. Of course, the underlying mechanisms by which growth occurs in real biological systems is much more complicated than the simplified scenario sketched above. For example, the amount of growth factors and their efficiency, which could be linked to some genetical predisposition, probably influences the overall rate of growth. Moreover, the other chemical processes involved with growth within a cell are also surely very important. For instance, the mechanism by which hypertrophy, rather than hyperplasia, takes place have been omitted here too. The only assumption we made is that the stimulus for growth to start is stress-related and that the response of the biological tissue to an external disturbance in the stress, i.e., to grow, should also be linked to the availability of biochemical energy (in the form of, for example, nutrients) within the tissue. In other words, if there is not enough biochemical energy available, the cells will be unable to duplicate at the "optimal rate", thus affecting the macroscopic growth rate.

The main result of the present work is that, under precise but general assumptions, the dynamics of growth in time are regulated, in the simplest case, by the thermodynamically admissible Eq. (3.12). The expected behavior in time can be of two types. If there is a large availability of nutrients (say $c \gg c_o$), the first term on the right-hand side of Eq. (3.12) tends to a constant, thus leading back to the homeostatic tensional state argument suggested in [1]. Conversely, for low nutrient concentration ($c \ll c_o$), the growth rate reduces linearly. It is worth emphasizing that the switch between growth and resorption is driven solely by the tensional state and is independent of the value of c.

While the simplifications of the model are self-evident (one-component medium, hyperelasticity, one chemical component), the link between mechanics and chemical transport are for the first time obtained on the basis of a precise thermomechanical basis, in which two main actors regulate growth: soluble factors (nutrients) and mechanical tension. No additional balance equations are introduced out of the standard ones: mass, momenta and energy balances. Simple admissible constitutive equations point out the coupled role of energy and tension in growth.

Let us note that, while for many authors the mixture theory seems to be a natural framework for growth mechanics, in the present work, we considered the material to be modelled by a single-component continuum. The well known difficulties in stating boundary value problems in a mixture theory can be circumvented by recent approaches [6]. While a multiple-components framework does not provide much better insights of the kinematics of growth [5], it is physically more consistent. That is, mass production as stated in (1.1) of the present paper would surely have more physical sense if interpreted as mass exchange between components of a mixture.

As a matter of fact, it is possible to rephrase the content of the present paper in terms of a two-phases reacting mixture, just one component providing a tensional contribution, the other being carrier of chemical energy. However, the key non-standard questions addressed in the present work, would remain in essence the same in a much more general multiphase context (see for instance [2]).

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